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O0286 *Klebsiella pneumoniae* KPC producer resistant to ceftazidime-avibactam due to a deletion in the *bla*KPC gene

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Background: *Klebsiella pneumoniae* producing blaKPC is a great health concern. Therapy with ceftazidime-avibactam represent a choice for the treatment of infections supported by these strains. We described an insurgence of resistance to ceftazidime-avibactam in an infected patient by a deletion of 6 nucleotides in the KPC gene sequence

Materials/methods: two strains, namely AMP920 and AMP2009 were isolated from same patient a month away. Antimicrobial susceptibility was performed both by broth microdiluition and Etest. Immunoenzymatic assay to detect carbapenemase was performed for both strains. KPC gene of both strains were amplified by PCR and sequenced. Enzyme activity towards carbapenems was tested by CarbaNP test and hydrolysis spectrophotometer assay.

Results: The two isolates differ in antimicrobial susceptibility testing. AMP920 show meropenem and imipenem resistance (MIC 32 and 32 mg/ml, namely). A month later carbapenems MIC decreased to 8 and 1 mg/ml, while ceftazidime-avibactam MIC increase from 1 to 16 mg/ml. Both isolates showed a positive immunocromathographic test for KPC enzyme, but only AMP920 showed positive CarbaNP test hydrolyzing imipenem. BlaKPC gene was ampliefied in both strains. After sequencing the two amplicons showed a KPC38 variant. The gene of second isolate showed a deletion of 6 nucleotides in position 498-503, bringing a mutate variant with the following deletion of a glutamic acid and leucine in position 167 and 168

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Conclusions: we register an insurgence of ceftazidime-avibactam resistance with decreasing activity against carbapenems in a *K. pneumoniae* strains. The resistance to ceftazidime-avibactam is due to a deletion of 6 nucleotides in position498-503, that bring to a new variant.